TO THE EDITOR: Resting bradycardia is an unquestionable marker of remodeling of the sinoatrial node rather than high vagal tone?

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A STALKING HORSE FOR RESTING HEART RATE IN ATHLETES

TO THE EDITOR: The paper by Boyett et al. (3) makes an argument in a theoretical analysis for a decrease in intrinsic pacemaker rate. This idea is not new. Boyett et al. (3) provide some further “food for thought.” I have some criticisms of this analysis. It is poor science to cross-compare data between the studies in parallel groups 2–11 (Table 1) that are not biologically similar. This is only valid in a longitudinal study such as one in which the conclusion could be different to that of Boyett’s. The arguments take no account of changes in the vagal mediated baro-heart rate reflex sensitivity following training in animal and human that also have been shown to be protective against ventricular fibrillation (5). Appraisal of heart rate variability (HVR) is superficial and there is no comment on its vagal dependence. Al-Ani et al. (1) based conclusions on several indices and did not use SDNN. Their data of HF peak change in the intrinsic heart rate in athletes and favoring the high cardiac vagal tone hypothesis: 1) little or no change in the intrinsic heart rate in athletes and 2) an increase in heart rate variability in athletes. Although authors suggest that intrinsic remodeling of the sinoatrial node would play a major role in the bradycardia seen in athletes, we believe that the limitations of the currently employed techniques, such as drug-induced autonomic blockade with propranolol and atropine as well as heart rate variability, for indirect measuring cardiac vagal activity, compromised the conclusions and could lead to misassumptions. Therefore, we believe that combining direct recording of the cardiac vagal tone in athletes and/or experimental animals to cellular approaches ranging from enzymes and other cellular mediators to electrophysiological properties of the sinoatrial none cells would help to unravel the precise role played by the parasympathetic activity and by the cells in the sinoatrial node in producing resting bradycardia in trained athletes. However, there is still need for refining the experimental approaches to investigate such mechanisms.

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of pacemaker currents but the influence of autonomic innervation should not be dismissed.

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COMMENT ON “IS THE RESTING BRADYCARDIA IN ATHLETES THE RESULT OF REMODELING OF THE SINOATRIAL NODE RATHER THAN HIGH VAGAL TONE?”

TO THE EDITOR: The basal heart rate (HR) has always been considered an indirect index of fitness, often used as a parameter in individuals undergoing physical training. However, where was discussed the origin of prominent bradycardia at rest observed in trained individuals, especially in high level athletes? The study by Boyett et al. (1) makes a systematic review of the findings of several authors, both clinical and experimental, and discusses the possible mechanisms responsible for bradycardia, with a focus on cardiac autonomic control and reduction of intrinsic HR (iHR). In fact, the autonomic nervous system has been touted as a major contributor to the bradycardia at rest. This bradycardia was due to adjustments in cardiac autonomic balance characterized by increased vagal autonomic drive and/or reduction of sympathetic autonomic drive (2). In this case, these adjustments may result from central or peripheral adaptations of structures involved with autonomic nervous system, i.e., afferent, central nuclei, efferent, and cardiac autonomic receptors. However, in experimental studies with rats, the reduction in basal HR, due to the physical training, seems to depend on the two mechanisms, i.e., reduction in iHR and adaptations in cardiac autonomic balance (3, 4). Moreover, the study of Tezini et al. (4) also shows that the bradycardia at rest in young animals is more dependent on iHR reduction, whereas in older animals it is more dependent on the autonomic balance adjustments. Finally, studies in humans are needed to confirm these findings.

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